

METHODS

On-Line Epicardial Mapping of Intraoperative Ventricular Arrhythmias: Initial Clinical Experience

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An on-line automatic mapping system was developed for beat by beat display of epicardial activation during ventricular tachycardia induced at the time of cardiac surgery. A sock array of 110 button electrodes was used to record and display local activation on a video monitor at 8.3 ms intervals. On instant replay in slow motion, epicardial pacing sites were accurately localized to the nearest electrode. Local unipolar electrograms were also recorded, first from the sock array, then from an array of 16 transmural needle electrodes. The epicardial display was verified by retrospective manually derived maps using the recorded epicardial electrograms.

In four patients with coronary artery disease and recurrent inducible ventricular tachycardia, earliest epicardial activation was located on slow motion replay

within 1 minute. Subendocardial sites of early activation were located within 10 minutes by replay of electrograms from the needle array before ventriculotomy. Transmural and endocardial resection of these sites prevented inducibility of the tachycardia on postoperative electrophysiologic study in three of the four patients. There has been no clinical recurrence of ventricular tachycardia after 3 to 14 months of follow-up despite cessation of antiarrhythmic therapy in three of the patients.

This technique has unique advantages over existing mapping methods. It provides beat by beat display of activation sequences so that clinical tachycardias that are short in duration or pleomorphic in configuration now become amenable to mapping. In addition, it markedly shortens total time on cardiopulmonary bypass.

In the past, surgery for the treatment of ventricular tachycardia has included resection of ventricular aneurysm with or without aortocoronary bypass grafting (1-4). With these undirected surgical procedures, success in controlling the arrhythmia has been poor (5,6). In recent years with the advent of ventricular mapping techniques, the results of surgery have improved as surgeons have taken a more direct approach. Now with identification of the site of earliest myocardial activation during ventricular tachycardia, techniques have been developed that excise (5,7) or electrically isolate (8,9) this area from surrounding myocardium.

The technique used for intraoperative mapping of ventricular tachycardias has not substantially changed from that originally used for determining ventricular activation in the Wolff-Parkinson-White syndrome (10,11). A hand-held probe is generally used to record local unipolar or bipolar electrograms sequentially at 50 to 70 epicardial sites. Local

activation times are measured relative to a fixed reference electrogram and the data are then collated to produce an activation map. In experienced hands, recordings take 5 to 15 minutes to collect and an additional 20 to 30 minutes are required to derive the map (12), thus increasing the duration of normothermic cardiopulmonary bypass by at least 30 to 60 minutes (13). In addition, this traditional technique has other limitations. Most important among these is that it requires the tachycardia to be sustained long enough for the data to be collected. Ventricular tachycardias of short duration or of a polymorphic and unstable nature cannot be mapped.

Recent advances in electronics have allowed the development of new techniques that can simultaneously record a large number of electrograms (14-17). At present these techniques have been largely confined to experimental studies, although one report (18) describes a computerized mapping method used successfully in the clinical intraoperative setting. We have reported an on-line analog approach to mapping experimental arrhythmias (19). Since then, the method has been modified to meet the requirements of clinical intraoperative mapping. This report describes the initial experience with this approach in four patients in whom the

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mean time to visualize an accurate intraoperative epicardial map was 1 minute from the time of initiation of a tachycardia.

Methods

The recording system comprises two units working in parallel. One provides a recording of all the analog signals in the form of unipolar local electrograms that are stored on a video recorder (20). The second provides an on-line analysis and video display of activation, which is stored on the second video recorder to provide instant replay of the temporal sequence of excitation (19). Both systems are linked by a common time code.

Analog recording system. One hundred ten unipolar local electrograms are time-division multiplexed to generate a single video signal that can be recorded on a video cassette tape recorder. Each local electrogram is sampled at a rate of 960/s and provides a single bandwidth of 170 Hz (3 dB). A demultiplexer unit reconstructs each signal with sample and hold amplifiers. Active filters provide intersample smoothing and ensure accurate signal reconstruction free from aliasing and errors of commission.

The stored signals were replayed on a Mingograph ink writer to provide an off-line record of the electrograms and

the time code. These off-line records were used to provide manually generated epicardial activation maps for retrospective comparison with the on-line video display of epicardial activation used at the time of surgery. Local activation was defined by the dominant negative deflection at the isoelectric line, and activation times were measured from a common time reference provided by the time code. At a paper speed of 250 mm/s, the measurements were accurate to ± 1 mm, thus providing an accuracy of activation times of ± 4 ms.

On-line activation display. The demultiplexer unit also provided reconstructed but unfiltered local electrograms to an on-line activation analyzer. This unit utilizes local electrograms to provide intensity signals at the time of local activation. This is accomplished by differentiation of the local electrograms and holding the values for peak negative dV/dt as intensity signals that brighten synchronously with local activation. A video signal generator then assembles all the intensity signals into one single-video format signal that is displayed on a raster scan monitor. The resulting image representing the array of epicardial electrodes consists of a matrix of dots that brighten at the time of local activation. The threshold for display of activation was adjustable from -1 to -5 V/s. This display system provided 120 video fields/s, which allows a temporal resolution of the activation sequence of 8.3 ms. The activation video image was recorded on a second video recorder that had the facility of instant replay during surgery, allowing the epicardial activation sequence to be analyzed in slow motion on a frame by frame basis. Each frame also displayed the common time code in binary form so that it could be temporally related to the analog recordings.

Recording electrodes. To record global epicardial activation, a sock array of button electrodes (12) was used. One hundred ten unipolar button electrodes were positioned in a standardized pattern within the mesh of a nylon sock

Figure 1. Derivation of epicardial activation display. **Panel A** shows the heart depicted in a polar projection with the apex at the center and the base at the circumference. The left anterior descending artery (LAD) runs in a 12 o'clock position. **Panel B** shows the distribution of the 110 button electrodes over the epicardial surface of the heart. The index row of electrodes A_1 to A_{10} was always positioned just to the left of the left anterior descending artery. **Panel C** shows the television monitor display that would be seen if all the electrodes simultaneously detected local activation.

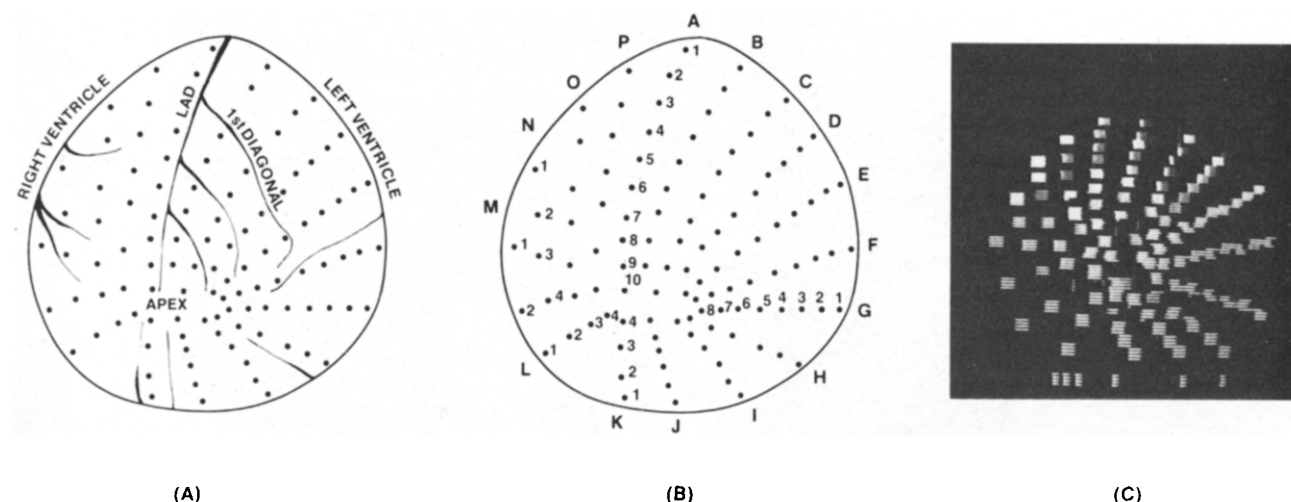


Table 1. Summary of Findings in the Four Patients With Intraoperative Mapping*

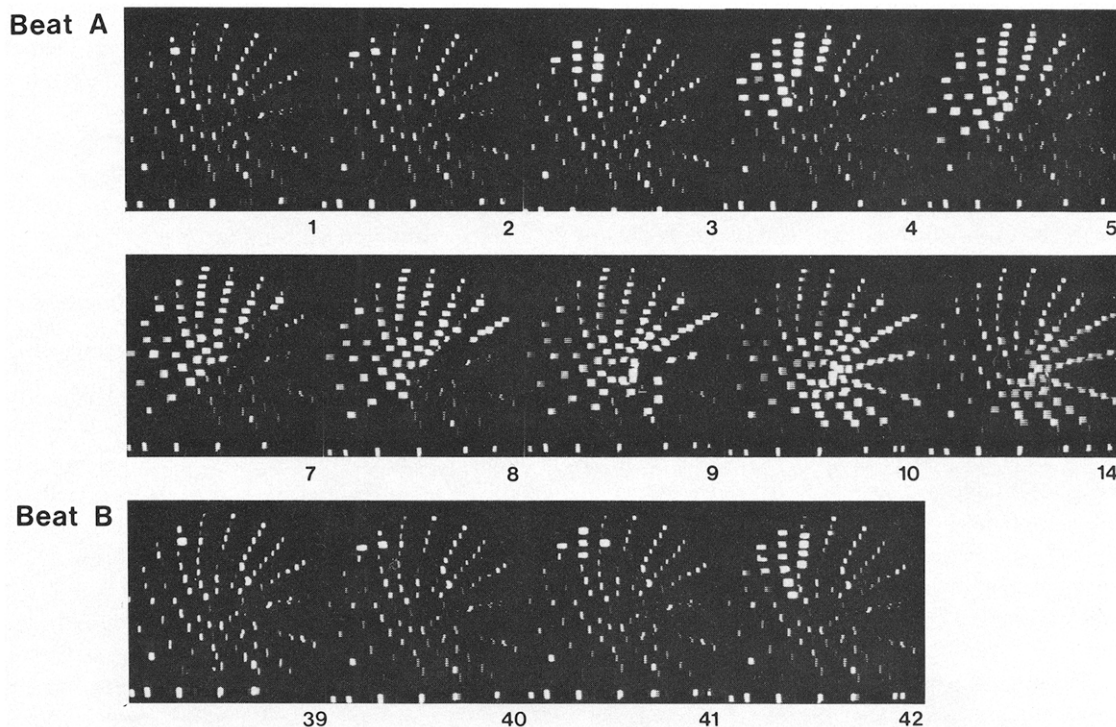
| Case | Age (yr) | Diagnosis | EF (%) | LVEDP (mm Hg) | Medications | Spontaneous Arrhythmia | Induced Arrhythmia† | Mode of Termination |
|------|----------|------------------|----------|---------------|---|------------------------------|---|------------------------------|
| 1 | 53 | CAD, LV aneurysm | 20 to 39 | 26 to 31 | Quinidine, nifedipine | Monoform VT at 195/min | S ₂ S ₃ , identical configuration | Burst pacing |
| 2 | 54 | CAD, LV aneurysm | 21 | 18 | Quinidine, procainamide, amiodarone | Monoform VT at 190/min, VFib | S ₂ S ₃ , two configurations | DC shock |
| 3 | 55 | CAD, LV aneurysm | — | 11 | Procainamide, disopyramide, quinidine, verapamil, amiodarone, bretylium | Nonsustained, polyform VT | S ₂ S ₃ S ₄ S ₅ , one sustained configuration | DC shock |
| 4 | 44 | CAD, LV aneurysm | < 20 | 28 | Procainamide, quinidine, mexiletine, amiodarone, encainide | Monoform VT at 225/min | S ₂ S ₃ , three configurations | Programmed stimuli, DC shock |

*All patients had angiographic appearance of anterior left ventricular aneurysm. †Indicates the number of premature stimuli (from S₂ to S₄) required to initiate the tachycardia. CAD = coronary artery disease; DC = direct current; EF = ejection fraction; LV = left ventricular; LVEDP = left ventricular end-diastolic pressure; V Fib = ventricular fibrillation; VT = ventricular tachycardia.

that was tailored to the dimensions of each individual patient's heart. The button electrodes consisted of a brass pin, 1.5 mm in diameter, set in a Teflon disc, 8 mm in diameter. Multistranded stainless steel insulated wire connected the button electrodes to the multiplexer unit.

The standard array of epicardial electrodes is shown in Figure 1. The heart is shown in a polar projection with the

Figure 2. Video display of epicardial activation during epicardial pacing in a canine heart. A hand-held probe was used to pace the heart from a site adjacent to electrode P₂ on the right ventricular outflow tract. A numbered sequence of video images (fields) is shown, each representing 8.3 ms. Video fields 1 and 39 show the earliest activation of two sequential paced beats A and B. In both cases, the activation is correctly shown to start at electrode P₂ (the pacing site). See text for further explanation.



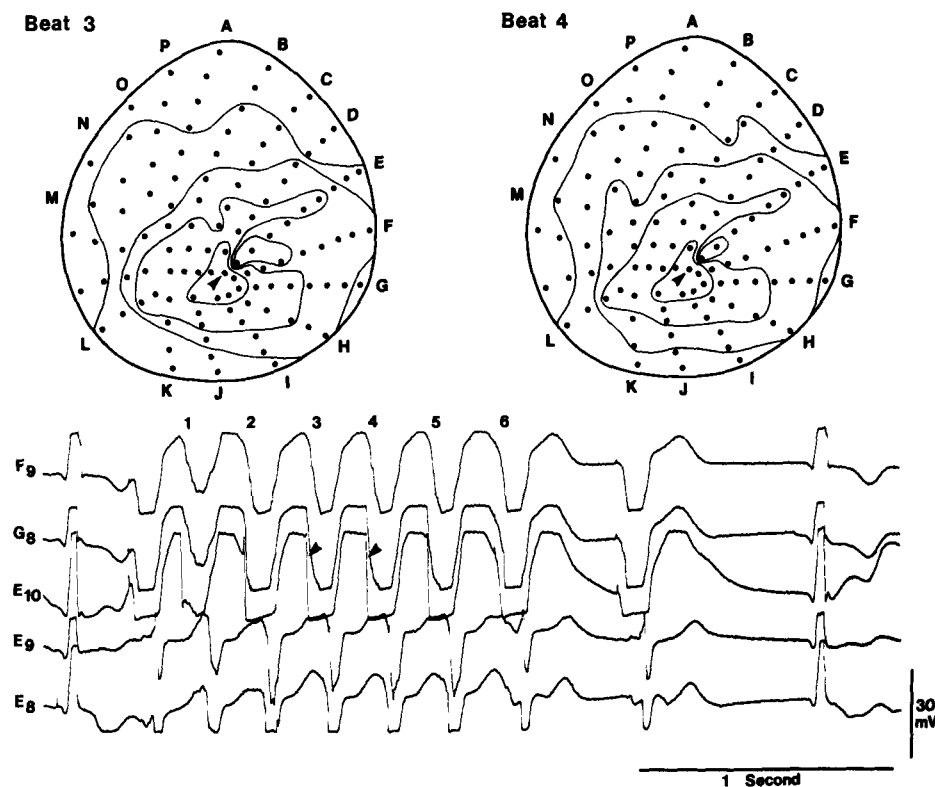


Figure 3. Case 1. **Lower panel** shows a pacing-induced six beat salvo of ventricular tachycardia recorded from five neighboring epicardial electrodes in the sock array. Earliest (arrowheads) and latest activation during the salvo occurred at electrodes E_{10} and E_9 , respectively, and were situated over the left ventricular apex. **Upper panel** shows the global epicardial activation for beats 3 and 4 of the salvo of ventricular tachycardia in the **lower panel**. These maps were derived from off-line manual measurements of all the local electrograms and illustrate the spread of activation in 30 ms isochrones. **Arrowheads** indicate that earliest origin of both beats was at electrode E_{10} and latest at E_9 .

apex at the center and the base around the circumference. The indexing row of electrodes A1 to A10 is orientated along the course of the left anterior descending coronary artery. Rows L, M, N, O and P are situated over the right ventricle. The automatic video display of epicardial excitation shows "activated" electrodes in the same polar projection of the heart.

To determine transmural activation, up to 16 multipolar needle electrodes were used. Each needle was made of 21 gauge steel and had 8 tungsten wire electrodes set at 3 mm intervals (21). These needle electrodes were not positioned in any fixed array, but rather distributed at 2 to 3 cm intervals from each other as best indicated by the results of the preceding epicardial map. Consequently, only analog local electrograms from the subepicardium to the subendocardium were recorded, and there was no attempt at automatic display of transmural activation.

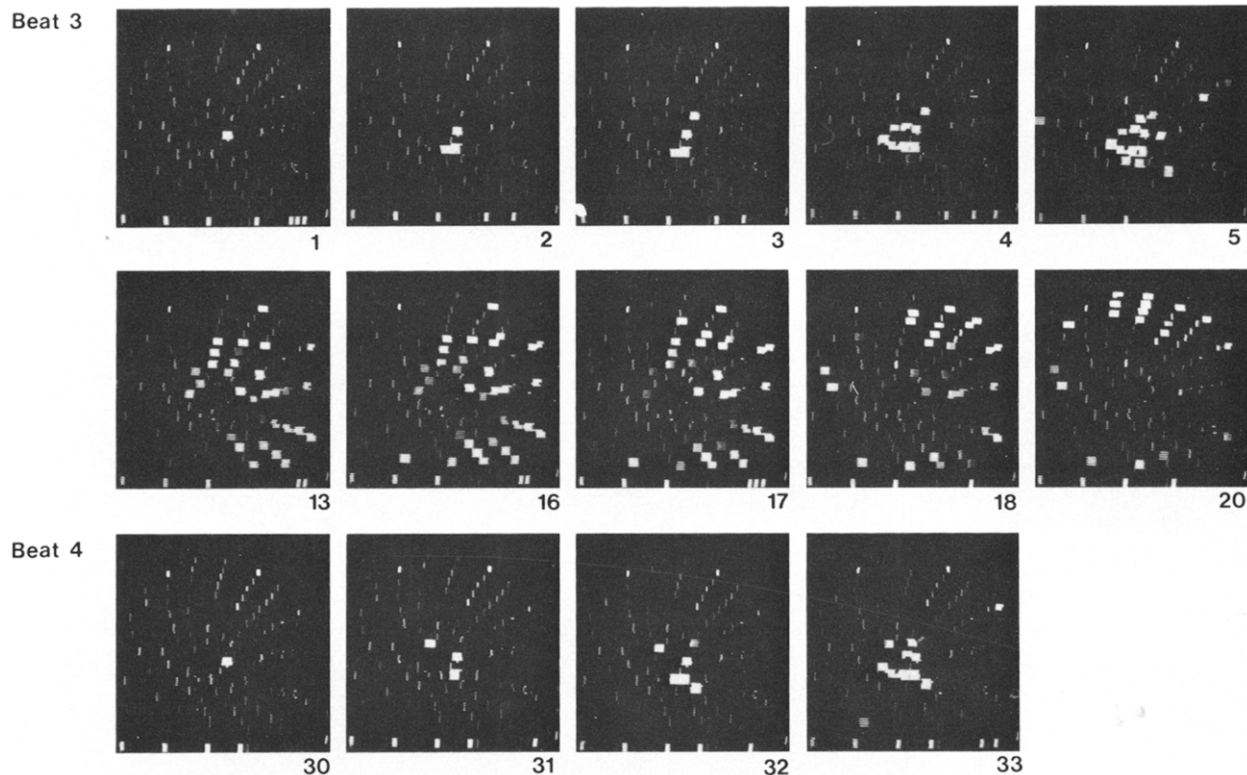
All local electrograms were recorded in the unipolar mode with a reference electrode attached to the diaphragm. The electrodes, the buffer amplifiers and the multiplexer unit were electrically isolated from the remainder of the recording equipment by a radio frequency transformer. The multiplexer was battery driven and transmitted the multiplexed analog data as a video-modulated radiofrequency signal which was transmitted by shielded cable and demodulated by a TV tuner.

Patient selection. Patients with symptomatic coronary artery disease and recurrent episodes of ventricular tachy-

cardia were considered for intraoperative mapping. All patients underwent routine hemodynamic, angiographic and intracardiac electrophysiologic investigations. The four patients chosen for surgery either had ventricular tachycardia that was refractory to medical therapy or required revascularization surgery for control of angina (Table 1). Informed consent was obtained for the pre- and postoperative investigations and for the intraoperative mapping and surgical procedures.

Preoperative electrophysiologic studies were performed using programmed electrical stimulation from the right ventricular apex or outflow tract or the left ventricular apex. Stimuli used were twice the late diastolic threshold strength and 2 ms in duration. After a basic train of eight stimuli at cycle lengths of 600, 500 or 400 ms, up to four programmed premature stimuli were used to induce ventricular tachycardia. In one patient, endocardial catheter mapping was performed during tachycardia. Induced episodes of ventricular tachycardia were terminated by burst pacing or overpacing with programmed premature stimuli.

Intraoperative procedure. With the patient on normothermic cardiopulmonary bypass but without cross-clamping of the aorta, the sock electrode array was applied. Contact between the electrodes and the epicardial surface of the heart was maintained by adjusting the tension of a suture within the mouth of the sock situated over the atrio-ventricular ring. Electrical contact was further facilitated by moistening the epicardial surface with saline solution. The



video monitor display was then optimized by adjusting a common gain control to maximize the number of sites showing activation without displaying artifactual effects caused by excessive gain. Electrode sites over thick epicardial fat layers and over myocardial scar failed to be displayed. Occasionally movement artifact at one electrode site produced spurious activation on the video display. This situation could be readily recognized by inappropriate flickering of the site in question; if stability could not be obtained by adjustment of the sock, that electrode was subsequently ignored.

Ventricular stimulation was applied through a pacing catheter positioned in the right ventricular apex. Programmed stimulation sequences similar to that which had been successful in inducing ventricular tachycardia preoperatively were then applied. After 30 to 60 seconds of the tachycardia was recorded on both the analog and automatic display recorders, the arrhythmia was terminated and a slow motion frame by frame review was made of the automatic display. The sites of earliest epicardial activation or the region encompassed by a recirculating wavefront of activation were then marked on the heart by sutures and the sock was removed.

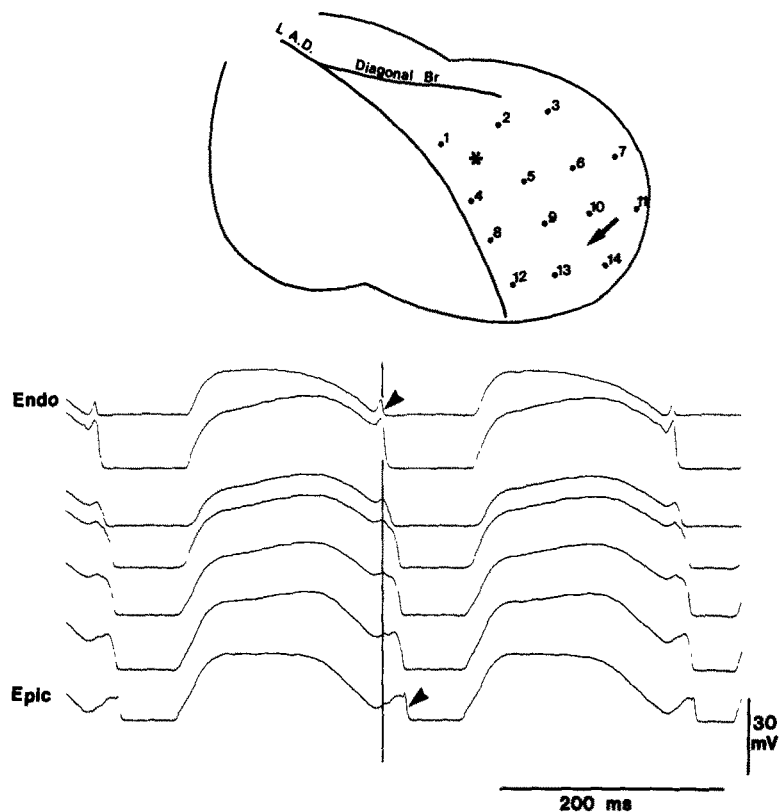
Sixteen transmural needles each with eight electrodes were then distributed so as to broadly encompass the key epicardial sites identified by the sock array. Their relative positions to each other and major anatomic sites were recorded by sketch. After an interval of approximately 5 min-

Figure 4. Case 1. On-line video display of the same beats 3 and 4 shown in Figure 3. These were instantly available on replay at the time of surgery. Each panel represents an 8.3 ms time field, and the numbers under each panel represent the number of that field with respect to the first one to show any activation. The video display depicts the electrode array in the same polar projection shown in Figure 1. The **extreme left top and bottom panels** show that activation began each time at electrode E₁₀. Subsequent spread closely parallels the sequences shown in Figure 3. Failure of some right ventricular electrodes to show activation was due to the presence of thick epicardial fat deposits in those locations.

utes, to allow injury currents to subside, the tachycardia was reinduced and a short recording was made of the analog epicardial, intramural and subendocardial electrograms. Replays of these electrograms were analyzed and the region of earliest activation (referenced to the common time code) could be identified within 10 minutes of making the recording.

On the basis of this information, a ventriculotomy was made close to the site of earliest activation. Our original intention had been to confirm the site of earliest endocardial activation with the use of an endocardial probe. However, in all four patients, once the ventricular incision had been made we were unable to reinduce the arrhythmia. After the mapping procedure had been completed, cardioplegia was instituted and aortocoronary bypass grafting with or without aneurysm resection was carried out. In addition, in all four patients an additional area of endocardium was excised,

Figure 5. Case 1. **Upper panel** schematically shows an anterior view of the heart with the location of 14 octapolar needle electrodes as seen at operation. **Asterisk** indicates epicardial pacing site. **Arrow** indicates location of epicardial electrode E₁₀ at the site of earliest epicardial activation during tachycardia. **Lower panel** shows the transmural electrograms from needle 10 at 3 mm intervals from the subepicardium (Epic) (**lowest tracing**) to the subendocardium (Endo) (**top tracing**). Each beat of the tachycardia began at the subendocardium (**upper arrowhead**).



which included the site of origin of the arrhythmia as determined by mapping. At the end of the operation, before weaning from bypass, programmed electrical stimulation was repeated to assess the immediate electrophysiologic status.

Before the patient's discharge from the hospital 2 weeks later, a postoperative electrophysiologic study was performed in the catheterization laboratory. At that time, the same stimulation program that successfully induced tachycardia before operation was assessed. When no arrhythmia was induced, an additional premature stimulus was added. After discharge, the patients were followed up with 48 hour ambulatory electrocardiographic recordings at 1 week, 4 weeks, 3 months and every 4 months thereafter.

Experimental studies. Initially, the mapping was evaluated in the animal laboratory. In 12 separate in situ canine hearts, epicardial pacing was performed using a hand-held pacing probe. Different pacing sites were used in each heart with the sock array in place. An observer was always able to correctly detect the pacing site from the video display, which showed activation originating from the nearest button electrode in the sock. Figure 2 shows two sequentially paced beats (A and B) from one such experiment. The heart was paced from the right ventricular outflow tract adjacent to electrode P2. Each panel of the figure shows one video field representing 8.3 ms in time. The video fields are in numbered sequence, starting with field 1, which shows earliest activation of beat A. Field 39 shows the earliest activation

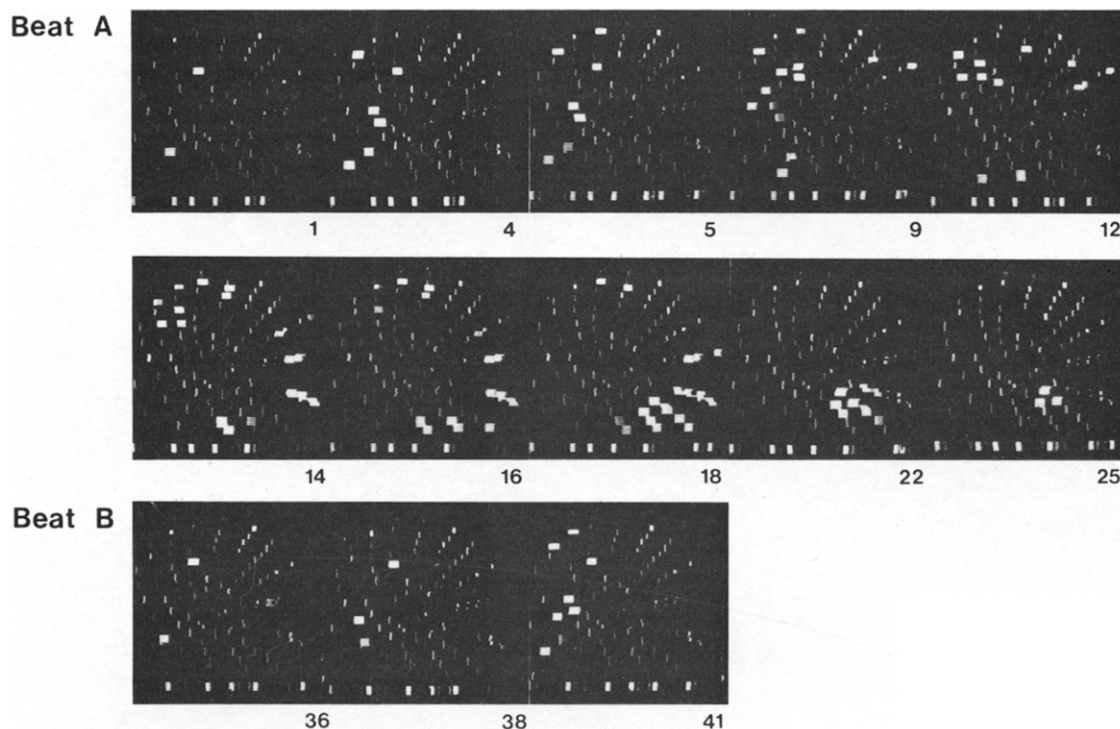
of beat B. Although the first two fields of beat B (29 and 40) are identical to that of beat A, subsequent fields (41 and 42) show some differences. Such differences in detail between two identical cardiac cycles were due to aliasing and were seen on both the automatic video display and the manually generated retrospective maps.

Results

Case 1

This patient had recurrent sustained episodes of monomorphic ventricular tachycardia at a rate of 195 beats/min associated with presyncope, severe angina and left ventricular failure. These spontaneous episodes required urgent electrical cardioversion. At electrophysiologic study in the catheterization laboratory, ventricular tachycardia of identical configuration to that of the spontaneous episodes could be readily induced with two programmed extrastimuli. These were terminated readily with burst pacing.

Intraoperative epicardial mapping. At the time of surgery during epicardial mapping within a self-imposed 10 minute time period allotted for induction of tachycardia, it was only possible to induce two 6 beat salvos of ventricular tachycardia (Fig. 3, lower panel). After a sinus beat, the fortuitous timing of a basic stimulus induced six beats of ventricular tachycardia that terminated spontaneously and was followed by a paced and a sinus beat in that order. The



video display of epicardial activation showed that the earliest epicardial activation of each beat of both salvos of ventricular tachycardia was at electrode E10. This was ascertained by slow motion replay at the time of operation within 1 minute of the induction of the salvos. Figure 4 shows selected still frames from that video display for beats 3 and 4 of the salvos shown in Figure 3. Retrospective manually generated maps, derived from analysis of the analog epicardial electrograms, confirmed electrode E10 as the site of earliest activation. These maps for the same beats 3 and 4 of the salvo are shown in 30 ms isochrones in the upper panel of Figure 3.

Intraoperative transmural mapping. An epicardial suture was used to mark the site of electrode E10, and the sock electrodes array was replaced by 14 transmural multipolar plunge electrodes that were distributed over the anterior aspect of the left ventricle (Fig. 5, upper panel). After an interval of 5 minutes, to allow injury currents to subside, programmed extrastimuli were again applied by a hand-held probe at the location marked by an asterisk shown in Figure 5. This time a sustained tachycardia was induced of an identical configuration to that of the spontaneous episodes. Replay and visual analysis of the transmural electrograms immediately after the induction of tachycardia revealed within 10 minutes that the earliest site of activation was at the subendocardial site of needle 10, which was adjacent to the site of earliest epicardial activation during the previous salvos of tachycardia. The transmural electrodes recorded during the sustained tachycardia from needle 10 are shown in

Figure 6. Case 2. Video display of epicardial activation of two sequential beats of ventricular tachycardia. Each beat began simultaneously at two sites (A_5 and L_2) at the anterior and inferior borders of an extensive apical aneurysm (panels 1 and 36). There were no viable myocardial fibers in the aneurysm. Consequently, electrodes over the apical region fail to display any local activation.

the lower panel of Figure 5. Unfortunately, it was not possible to collate the epicardial and transmural data because of the differing natures of the tachycardias. Therefore, we were unable to say whether the tachycardia was due to a small transmural circuit of reentry or had a monofocal origin in the vicinity of needle 10 and epicardial electrode E10. The delay of epicardial activation of 88 ms between electrodes E10 and E9 (only 1 cm apart) suggests reentry as a possible mechanism. A ventriculotomy of 3 cm was made in this location to perform endocardial mapping, but the latter procedure was thwarted by inability to induce any tachycardia after the incision was made.

Surgical procedure and clinical results. Gross inspection at the time of surgery revealed that in the dyskinetic area of the left ventricle, there was viable muscle interspersed with scar. Within the large area of dyskinesia, a full thickness resection, 2 × 3 cm, encompassing the site of earliest activation, was performed followed by slightly wider endocardial resection 2 to 3 mm thick. The patient also received three aortocoronary vein bypass grafts. At the end of the surgical procedure and 2 weeks before hospital discharge, it was no longer possible to induce ventricular

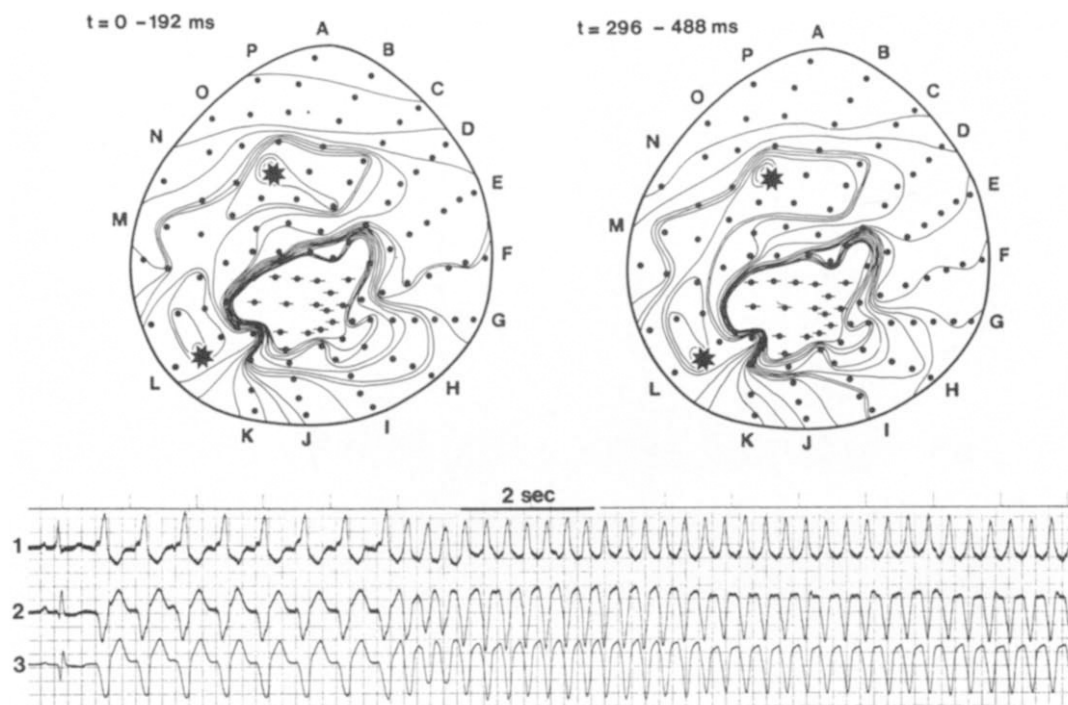


Figure 7. Case 2. **Upper panels** show retrospective manually derived epicardial activation maps of two sequential beats of the tachycardia seen in the **lower panel**. Activation is shown in 12 ms isochrones and begins simultaneously at sites A₅ and L₂ (asterisks), confirming the accuracy of the on-line display.

tachycardia by programmed pacing. The patient was discharged without antiarrhythmic therapy and during 14 months of follow-up study has had no recurrences symptomatically or on repeated Holter recordings.

Case 2

This patient had two spontaneous episodes of monomorphic ventricular tachycardia at a rate of 190 beats/min. Both episodes were associated with syncope and one degenerated into ventricular fibrillation during hospitalization. At electrophysiologic study, two programmed extrastimuli readily induced sustained ventricular tachycardia of identical configuration to that of the spontaneous episodes. Each episode could be terminated only by electrical cardioversion.

Intraoperative mapping. At operation, a large anterior left ventricular aneurysm was visualized. Epicardial mapping with the sock array revealed two sites of simultaneous epicardial breakthrough diametrically opposed at anterior and inferior borders of the aneurysm (Fig. 6). Epicardial sites on the scar tissue of the aneurysm understandably were not visualized on the video display of activation. Retrospective epicardial maps from the analog recordings of local electrograms confirmed the twin sites of earliest activation seen in the video display (Fig. 7). Transmural needle re-

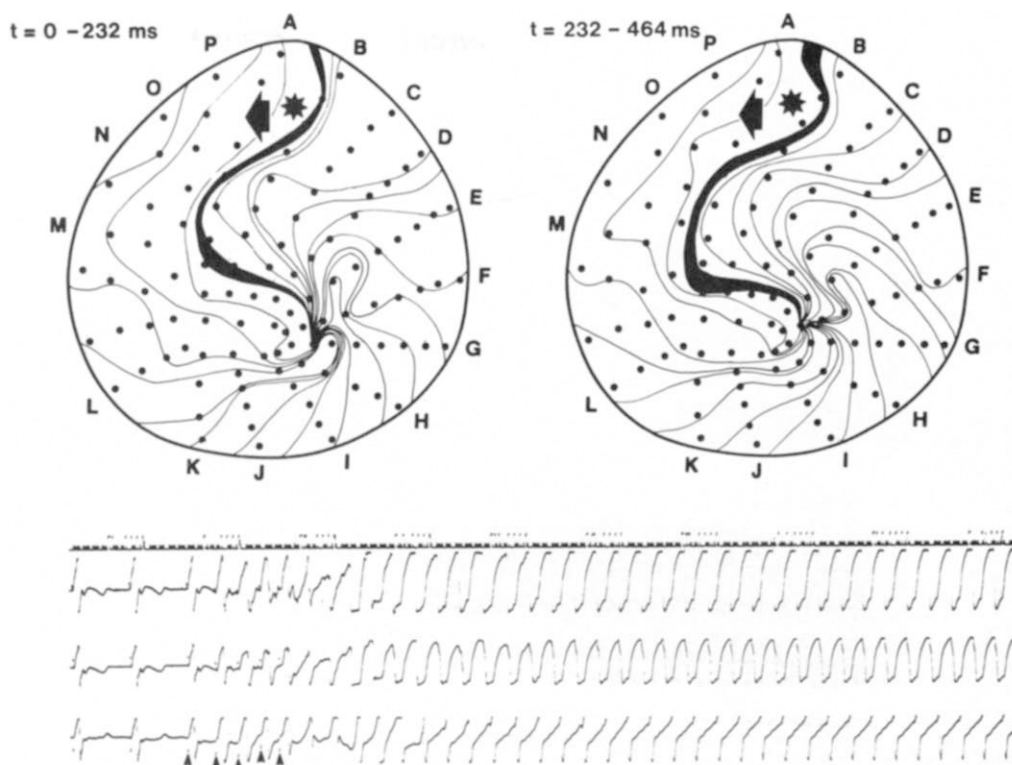
cordings examined at the time of operation confirmed these two locations and failed to reveal any greater detail of the activation sequence. Again, endocardial mapping could not be performed after the aneurysm was opened because of failure to induce tachycardia.

Surgical procedure and results. Aneurysmectomy was performed with resection of scarred endocardium along the margins of the aneurysm. This resection was more extensive in the areas identified by mapping. The patient also received triple vein bypass grafts. Repeat programmed stimulation both at the end of operation and 2 weeks before hospital discharge failed to induce ventricular tachycardia. The patient was discharged without antiarrhythmic drug therapy and has had no recurrences of the arrhythmia in a follow-up period of 8 months.

Case 3

This patient had frequent symptomatic runs of self-limited polymorphous ventricular tachycardia. Although he never had any documented episodes of spontaneous sustained ventricular tachycardia, at electrophysiologic study four programmed extrastimuli reproducibly induced a sustained monomorphic ventricular tachycardia at a rate of 180 beats/min (Fig. 8, lower panel).

Intraoperative mapping. The identical tachycardia was readily induced at operation, and the on-line display of epicardial activation showed, even in real time without slow motion replay, a continuous sequence of activation recirculating around a region of the left ventricular apex (Fig. 9). Retrospective epicardial maps confirmed the sequence



of continuous activity consistent with ventricular flutter (Fig. 8, upper panel). Transmural mapping with 16 plunge electrodes after removal of the sock array revealed that subendocardial activation at all the needle locations was secondary to epicardial activation (Fig. 10). Ventriculotomy prevented induction of the tachycardia so that endocardial mapping could not be performed. The angiographic appearance of the aneurysm was a result of dyskinetic ischemic myocardium.

Surgical procedure and results. A plaque scar 4×4 cm was found underlying the circle of epicardial activation and was resected together with a smaller full thickness portion of the left ventricle. The patient received two aorto-coronary bypass grafts. No ventricular arrhythmias could be induced at the end of operation or electrophysiologic study 2 weeks later. The patient was discharged receiving no antiarrhythmic therapy and there has been no recurrence of any ventricular arrhythmia symptomatically or on Holter electrocardiographic monitoring in 6 months of follow-up study.

Case 4

This patient had a 3 year history of recurrent ventricular tachycardia at a rate of 185 beats/min that was refractory to procainamide, quinidine, mexiletine, amiodarone and encainide administered either alone or in combination. At electrophysiologic study, two programmed extrastimuli readily induced sustained ventricular tachycardia of two configu-

Figure 8. Case 3. **Lower panel** shows three local epicardial electrograms in transition from programmed pacing (arrows) to sustained ventricular tachycardia. The trace above the top electrogram shows binary time code. **Upper panel** shows retrospective epicardial maps in 12 ms isochrones of the two sequential beats of the tachycardia seen below. Beginning with the isochrone marked by the asterisk and proceeding counterclockwise as indicated by the arrow, continuous epicardial activity was recorded from one cardiac cycle (extending from 0 to 232 ms) to the next (232 to 464 ms). The activation wheeled around the apex, although all the apical electrodes recorded local activation. The sequence is consistent with ventricular flutter.

rations, one of which was identical to that of the spontaneous episodes. Endocardial catheter mapping indicated that both types of tachycardia originated paraseptally on the high anterior portion of the left ventricular septum.

Intraoperative mapping. At operation, a true anterior left ventricular aneurysm was visualized. The video display of epicardial activation confirmed the results of catheter mapping and was, in turn, confirmed by retrospective manually generated maps. The sock was then replaced with needle electrodes, which were distributed around the region of the earliest epicardial activation. Unfortunately, programmed stimulation could only induce a ventricular tachycardia with a configuration not previously observed. This tachycardia originated in the subendocardium of the left ventricular wall close to the portion of the septum indicated

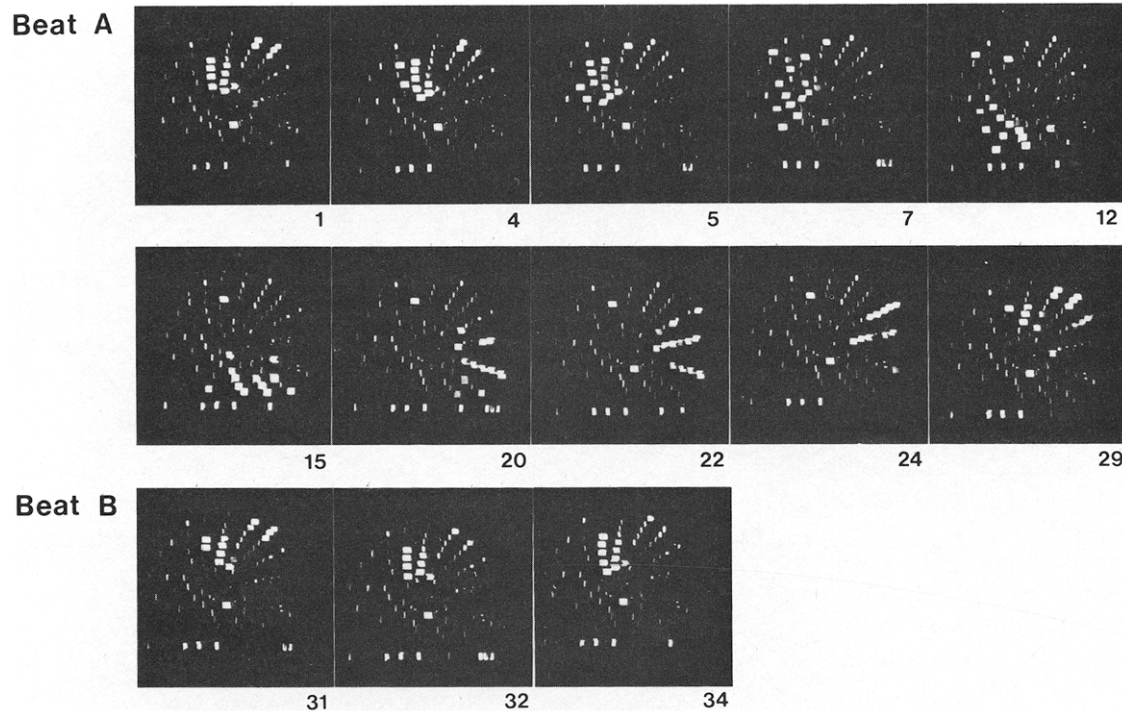


Figure 9. Case 3. On-line video monitor display of two sequential cardiac beats A and B showing continuous epicardial activation circulating around the apical region.

tachycardia of a configuration not previously seen. The patient was discharged on amiodarone, 600 mg daily, and there have been no symptomatic or documented ventricular arrhythmias in a short follow-up period of 3 months.

by the previous tachycardia as the earliest epicardial breakthrough. Opening of the aneurysm prevented further induction of tachycardia, and endocardial mapping could not be performed.

Surgical procedure and results. Aneurysmectomy together with wide endocardial resection extending over the high anterior portion of the left ventricular septum was performed. No aortocoronary grafting was feasible. At the end of operation, it was no longer possible to induce any ventricular arrhythmias. However, 2 weeks later electrophysiologic study with two premature stimuli did eventually induce, after prolonged testing, a sustained ventricular

Discussion

On-line mapping. To provide detailed maps of activation on a beat by beat basis, it is necessary to record simultaneously from a large number of electrodes. Several such recording systems have recently been developed and the results obtained with these systems in the field of experimental arrhythmias have been reported (14-17). The principal advantages they offer over traditional recording techniques are the capability of mapping single beats or short-lived arrhythmias, as well as polymorphous arrhythmias in which the activation sequence is constantly chang-

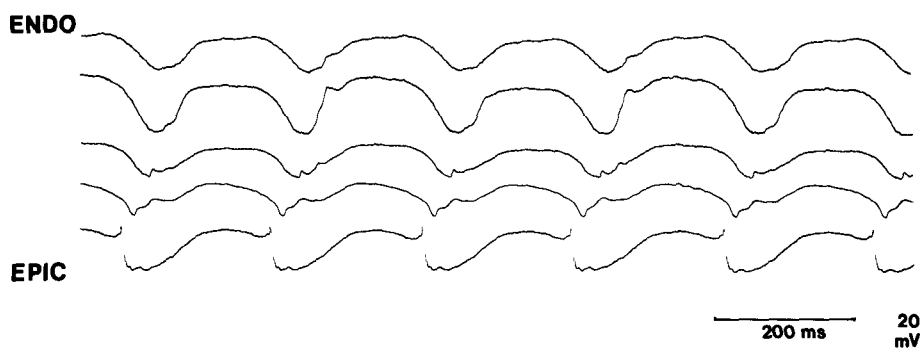


Figure 10. Case 3. Local unipolar electrograms from 1 to 16 transmural needles with electrodes set at 3 mm intervals. The **bottom tracing** shows that local activation from the subepicardium (EPIC) clearly leads all the other transmural potentials. Subepicardial activation preceded transmural and subendocardial (ENDO) activation at all 16 needle sites. See text for further explanation.

ing. Thus, events such as ischemia-induced ectopic beats (15,22) and the mode of onset of ventricular tachycardia and fibrillation (23,24) have become amenable to study. Because these methods still require considerable time for the analysis of recorded data, they are not directly applicable to clinical intraoperative mapping where time constraints are a severely limiting factor. A computer-based mapping system designed for the operative environment has been described (25), but reported clinical experience with this system has been limited to one case (18).

The present report describes the successful systematic clinical application of an on-line mapping system that provides a beat by beat display of epicardial activation sequences. The advantages offered by this approach include a significant reduction in time spent on cardiopulmonary bypass and the ability to map short salvos of ventricular tachycardia (as described for Case 1) that could not be mapped by traditional techniques. It also offers the opportunity to map polymorphous tachycardias, although the occasion for this has not arisen in the current experience. In its present form, because the technique is based on hard-wired analog circuitry, the on-line display of activation is restricted to a single projection of the epicardial electrode array and the only variables are a common gain control for the electrograms and a common sensitivity control for the activation display. In each of the cases studied, the accuracy of the automatic on-line display was retrospectively confirmed by manual maps derived from analysis of the analog signals recorded on a separate video recorder. This was best illustrated by Patient 2, who had two distinct sites of epicardial breakthrough at electrodes L₂ and A₅. Simultaneous replay of electrograms from both these sites showed that activation at A₅ preceded that at L₂ by 4 ms. Although Figure 6 shows that both these sites activated simultaneously, 75% of all the video fields displayed by this tachycardia showed A₅ preceded L₂ by one video field.

The automatic video display showed local activation only if local electrograms contained a downslope of at least 1 V/s. Analysis of the analog signals sometimes showed electrograms with an identifiable local activation despite a downslope of less than 1 V/s, and for this reason yielded more complete detail of local activation than the automatic display.

Value and limitations of epicardial and transmural mapping. The limitations of epicardial mapping of ventricular arrhythmias are well recognized (25,26). In an attempt to offset this limitation, we supplemented the epicardial information with subendocardial and transmural recordings from plunge electrodes. These were placed in what seemed an optimal disposition to include the epicardial sites of interest. This approach provided valuable data on events deep to the epicardium, which could only have been gained by endocardial mapping. The latter procedure is sometimes precluded by failure to induce ventricular tachycardia after opening the left ventricle (13,26) as was the

case in our four patients. Mapping with plunge electrodes, therefore, provides some safeguard against this common eventuality, and as a technique deserves to be routinely employed. It further provides an opportunity to gain sub-endocardial activation data in instances where extensive mural thrombi prevent traditional endocardial mapping (13). It does not, of course, obviate the need to obtain an endocardial map whenever possible.

Our current use of plunge electrodes requires approximately 10 minutes of cardiopulmonary bypass time for replay and analysis of the local electrograms. This time period could be significantly reduced by the future development of automatic on-line display modes of transmural activation. Our current experience with sixteen 21 gauge needle electrodes has not revealed any significant trauma or hemorrhage to the left ventricle, even when the site is in the posterior wall. One problem encountered was the difficulty in correlating the recording from the needles with that of the preceding sock array to produce comprehensive activation maps. This was, in part, due to time constraints preventing accurate placement of the needles with reference to the matrix of the sock electrode array and, in part, to the different configuration of the tachycardia induced after replacement of the sock by the needles (Case 4). In future studies, this limitation can be overcome by retaining the sock, placing the needles with reference to the sock array and expanding the recording capability of the system to its maximal capacity of 256 channels (20) so that both sets of electrodes can be simultaneously recorded.

Patterns of activation during ventricular tachycardia. To date, our observations are limited to patients with predominant left anterior descending coronary artery disease with the angiographic appearance of an anterior left ventricular aneurysm. Despite this commonality and the surface appearance of ventricular tachycardias that were similar in rate, the individual activation patterns were widely different. The tachycardia that occurred in Case 1 was monofocal in appearance on the epicardial map, but may have been caused by a small transmural circle of reentry as was suggested by the earliest activation occurring at the adjacent subendocardium. In contrast, the ventricular tachycardia induced in Case 3 was due to a much larger circle of reentry occurring predominantly at the epicardium with secondary activation of the subendocardium. This tachycardia was unique in displaying continuous epicardial activation from one cardiac cycle to the next. As such, it might be more accurately described as a ventricular flutter. Its appearance was very similar to the experimental atrial flutter caused by a leading circle of reentry described by Allesie et al. (27). The spontaneous polymorphic ventricular arrhythmias that this patient experienced and the polymorphic type of tachycardia that presaged the induced monofocal tachycardia (Fig. 8) suggest that some clinical ventricular arrhythmias of this type may also be due to ventricular flutter in which there is

a varying leading circle of reentry. Such observations made possible by the new techniques of clinical mapping hold the promise of important new insights into the mechanisms underlying human ventricular arrhythmias.

References

1. Buda AJ, Stinson EB, Harrison DC. Surgery for life-threatening ventricular tachyarrhythmias. *Am J Cardiol* 1979;44:1171-7.
2. Wald RW, Waxman MB, Corey PN, Gunstensen J, Goldman BS. Management of intractable ventricular tachyarrhythmias after myocardial infarction. *Am J Cardiol* 1979;44:329-38.
3. Gallagher JJ. Surgical treatment of arrhythmias: current status and future directions. *Am J Cardiol* 1978;41:1035-44.
4. Ecker RR, Mullins CB, Grammer JC. Control of intractable ventricular tachycardia by coronary revascularization. *Circulation* 1971;44:666-70.
5. Harken AH, Josephson ME, Horowitz LN. Surgical endocardial resection for the treatment of malignant ventricular tachycardia. *Ann Surg* 1979;190:456-60.
6. Harken AH, Horowitz LN, Josephson ME. Comparison of standard aneurysmectomy and aneurysmectomy with direct endocardial resection for the treatment of recurrent sustained ventricular tachycardia. *J Thorac Cardiovasc Surg* 1980;80:527-34.
7. Josephson ME, Harken AH, Horowitz LN. Endocardial excision: a new surgical technique for the treatment of recurrent ventricular tachycardia. *Circulation* 1979;60:1430-9.
8. Guiraudon G, Fontaine G, Frank R, Escande G, Etievant P, Carbol C. Encircling endocardial ventriculotomy: a new surgical treatment for life-threatening ventricular tachycardias resistant to medical treatment following myocardial infarction. *Ann Thorac Surg* 1978;26:438-44.
9. Ungerleider RM, Stanley TE, Lofland GK, et al. Electrophysiologic effects of encircling endocardial ventriculotomy for refractory ischemic ventricular tachycardia. *Surg Forum* 1980;31:291-3.
10. Cobb FR, Blumenschein SD, Sealy WC, Boineau JP, Wagner GS, Wallace AG. Successful surgical interruption of the bundle of Kent in a patient with Wolff-Parkinson-White syndrome. *Circulation* 1968;38:1018-29.
11. Sealy WC, Hattler BC, Blumenschein SD, Cobb FR. Surgical treatment of the Wolff-Parkinson-White syndrome. *Ann Thorac Surg* 1969;8:1-11.
12. Harrison L, Ideker RE, Smith WM, et al. The sock electrode array: a tool for determining global epicardial excitation during unstable arrhythmias. *PACE* 1980;3:531-40.
13. Josephson ME, Harken AH, Horowitz LN. Long-term results of endocardial resection for sustained ventricular tachycardia in coronary artery disease patients. *Am Heart J* 1982;104:51-7.
14. Wit LA, Allesie MA, Bonke FIM, Lammers W, Smeets J, Feghali JJ Jr. Electrophysiologic mapping to determine the mechanism of experimental ventricular tachycardia initiated by premature impulses. Experimental approach and initial results demonstrating re-entrant excitation. *Am J Cardiol* 1982;49:166-85.
15. Janse MJ, van Capelle FJL, Marsink H, et al. Flow of "injury" current and patterns of excitation during early ventricular arrhythmias in acute regional myocardial ischemia in isolated porcine and canine hearts. *Circ Res* 1980;47:151-65.
16. El-Sherif N, Smith AR, Evans K. Canine ventricular arrhythmias in the late myocardial infarction period. 8. Epicardial mapping of re-entrant circuits. *Circ Res* 1981;49:255-65.
17. Ideker RE, Smith WM, Wallace AG, et al. A computerized method for the rapid display of ventricular activation during the intraoperative study of arrhythmias. *Circulation* 1979;59:449-58.
18. Gallagher JJ, Kasell JH, Cox JL, Smith WM, Ideker RE. Techniques of intra-operative electrophysiological mapping. *Am J Cardiol* 1982;49:221-40.
19. Parson I, Mendler P, Downar E. On-line cardiac mapping: an analog approach using video and multiplexing techniques. *Am J Physiol (Heart Circ Physiol II)* 1982;242:H526-35.
20. Mendler P, Parson I, Downar E. Multichannel recording of cardiac potentials. *Med Biol Eng Comput* 1980;18:617-24.
21. Kasell J, Gallagher JJ. Construction of a multipolar needle electrode for activation study of the heart. *Am J Physiol* 1977;233:H312-7.
22. Downar E, Parson I. Mechanisms underlying ventricular arrhythmias of acute myocardial ischemia and reperfusion (abstr). *Circulation* 1981;64(suppl IV):IV-216.
23. Klein GJ, Ideker RE, Smith WM, et al. Epicardial mapping of the onset of ventricular tachycardia initiated by programmed stimulation in the canine heart with chronic infarction. *Circulation* 1979;60:1375-84.
24. Ideker RE, Klein GJ, Harrison L, et al. The transition to ventricular fibrillation induced by reperfusion after acute ischemia in the dog: a period of organized epicardial activation. *Circulation* 1981;63:1371-9.
25. Spielman SR, Michelson EL, Horowitz LN, Spear JF, Moore EN. The limitations of epicardial mapping as a guide to the surgical therapy of ventricular tachycardia. *Circulation* 1978;57:666-70.
26. Horowitz LN, Josephson ME, Harken AH. Epicardial and endocardial activation during sustained ventricular tachycardia in man. *Circulation* 1980;61:1227-38.
27. Allesie MA, Bonke FIM, Schopman FJG. Circus movement in rabbit atrial muscle as a mechanism of tachycardia. III. The "leading circle" concept: a new model of circus movement in cardiac tissue without the involvement of an anatomical obstacle. *Circ Res* 1977;41:9-18.